Reprinted from JOURNAL OF INVERTEBRATE PATHOLOGY, Volume 22, Number 2, September 1973

JOURNAL OF INVERTEBRATE PATHOLOGY **22**, 213–219 (1973)

Host Response in the White Shrimp, *Penaeus setiferus*, to Infection by the Larval Trypanorhynchid Cestode, *Prochristianella penaei*[1]

ALBERT K. SPARKS AND C. T. FONTAINE

National Marine Fisheries Service,
Gulf Coastal Fisheries Center,
Galveston Laboratory,
Galveston, Texas 77550

Received February 19, 1973

White shrimp, *Penaeus setiferus*, infected by the plerocercoid larva of the trypanorhynchid cestode, *Prochristianella penaei*, respond to infections in the heptaopancreas by developing a progressively denser cyst, composed of hemocytes, fibroblasts, and collagenlike fibers, around the parasite. The pleocercoid is eventually destroyed and resorbed leaving a dense fibrous capsule in the hepatopancreas. Worms in the adjacent hemocoel are encapsulated by a thin host cyst and a less intense infiltration of cells and deposition of fibers, insufficient to destroy the pleroceroid. It is believed that the resorption process explains the drop in intensity of infection in juvenile white shrimp after a certain size is attained.

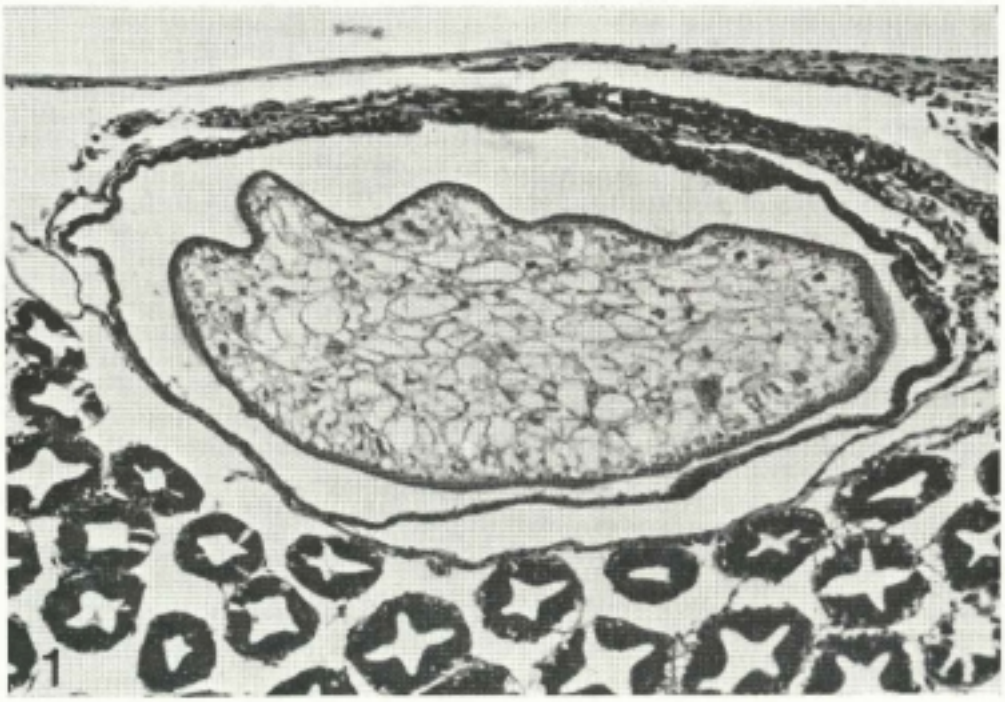
INTRODUCTION

A larval trypanorhynchid cestode was first reported from penacid shrimp, *Penaeus setiferus*, from Barataria Bay, Louisiana, by Sparks and Mackin (1957), but was not correctly identified. Kruse (1959) described *Prochristianella penaei* from the brown shrimp, *Penaeus aztecus*, the pink shrimp, *P. duorarum*, and the white shrimp, *P. setiferus*, and reported infection rates of 90.6, 97.0, and 94.4%, respectively, in the three species of shrimp from the northwest Gulf Coast of Florida. He also described *Parachristianella monomegacantha* and *Parachristianella dimegacantha* from *Penaeus duorarum*. Since only two specimens of *P. monomegacantha* and one of *P. dimegacantha* were found in his study and neither Hutton et al. (1959), Woodburn et al. (1957) nor Aldrich (1965) reported either species in Florida or Texas, it seems reasonable to assume that the vast majority of the heavy infections of penaeid shrimp by trypanorhynchid cestodes in the Gulf of Mexico are by *Prochristianella penaei*.

Aldrich (1965) conducted an ecological study of *Prochristianella penaei* infections in penacid shrimp in the Galveston Bay complex of Texas over a 3-year period. He found that individual brown shrimp were consistently more heavily parasitized than white shrimp of comparable size, although percentage was the same. Infections of both species were obviously obtained in the bays since both the incidence and intensity of infection increased as the shrimp increased in size. There is, however, a "leveling off" in intensity of infection in brown shrimp above 14 mm carapace length and a decline in white shrimp that Aldrich attributed to either a change in feeding or an increase in immunity.

Interestingly, apparently no one examined the pathological effect of *P. penaei* on the shrimp or the host's response to the

[1] Contribution No. 357, National Marine Fisheries Service, Gulf Coastal Fisheries Center, Galveston Laboratory, Galveston, Texas 77550.



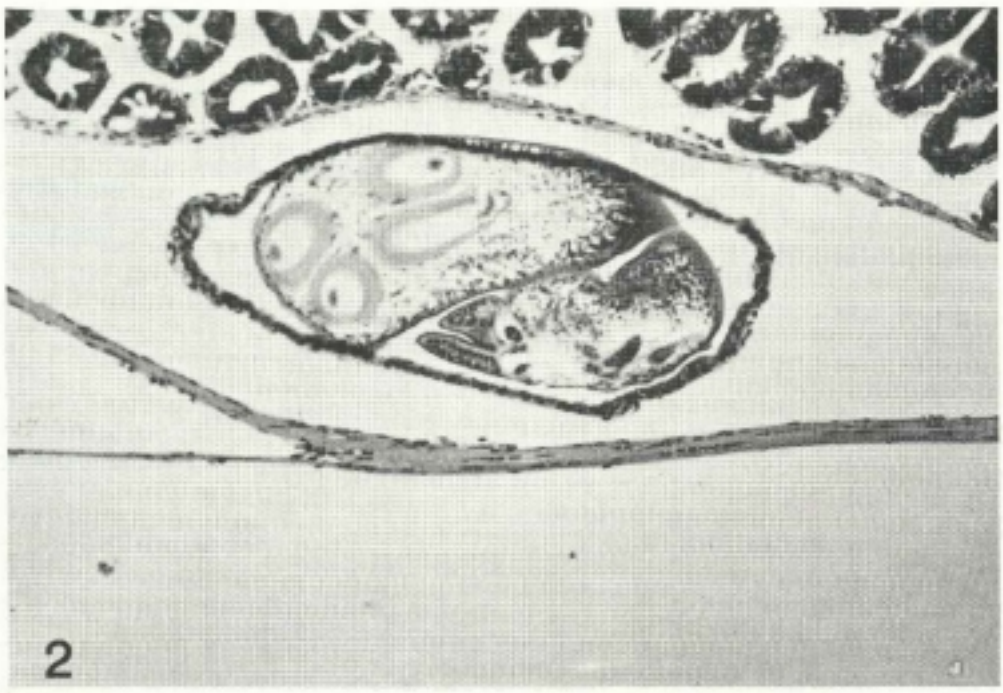


FIG. 1. Plerocercoid larva of *Prochristianella penaei* located between the acini of the outer portion of the hepatopancreas. H&E, ×175.

FIG. 2. Plerocercoid larva of *Prochristianella penaei* located just outside the hepatopancreas, separated from that organ by the connective tissue sheath that surrounds the hepatopancreas. H&E, ×175.

presence of one of the most common parasites of shrimp in the Gulf of Mexico.

MATERIALS AND METHODS

Ten live juvenile white shrimp were obtained from a bait dealer on Galveston Bay, transported alive to the Galveston Laboratory of the Gulf Coastal Fisheries Center, and examined grossly for the presence of the white spots in the hepatopancreas that are diagnostic of *P. penaei* infections. Those shrimp (6) obviously

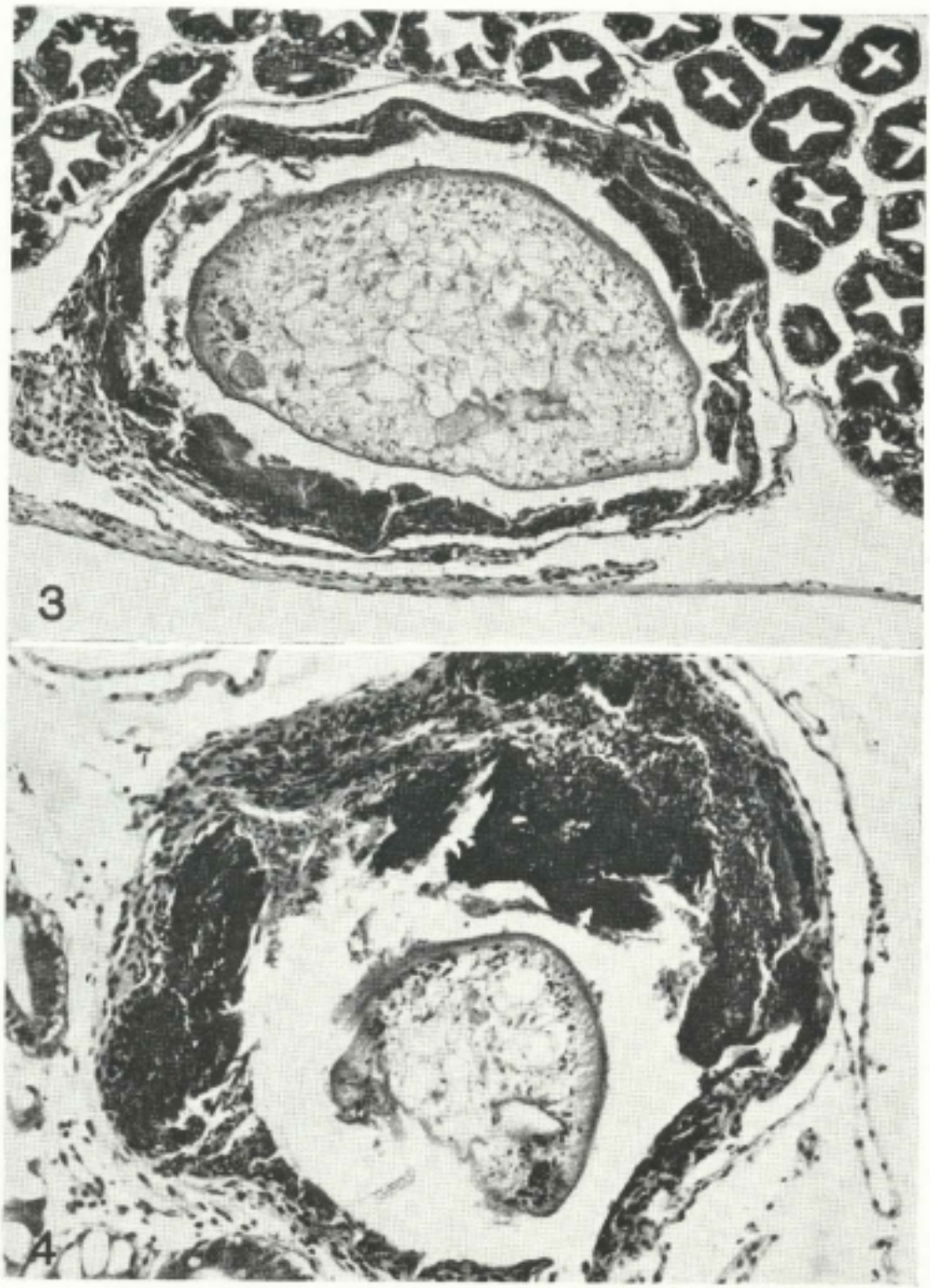


FIG. 3. Preliminary hemocytic response to plerocercoid larva in hepatopancreas. H&E, ×175.

FIG. 4. Thickened encapsulation with some fibroblasts appearing. H&E, ×220.

containing the cestode were fixed by hypodermic injection of 10% buffered formalin into the hepatopancreas, a necessity because of the rapid enzymatic autolysis of this organ postmortem. Sections were obtained by routine histological techniques and stained with either Harris's hematoxylin and eosin or Mallory's triple stain. Photomicrographs were made with a Zeiss Photomicroscope.[2]

RESULTS

The normal location of *P. penaei* in shrimp is between the acini of the outer

[2] The use of trade names in this publication does not imply endorsement of commercial products.

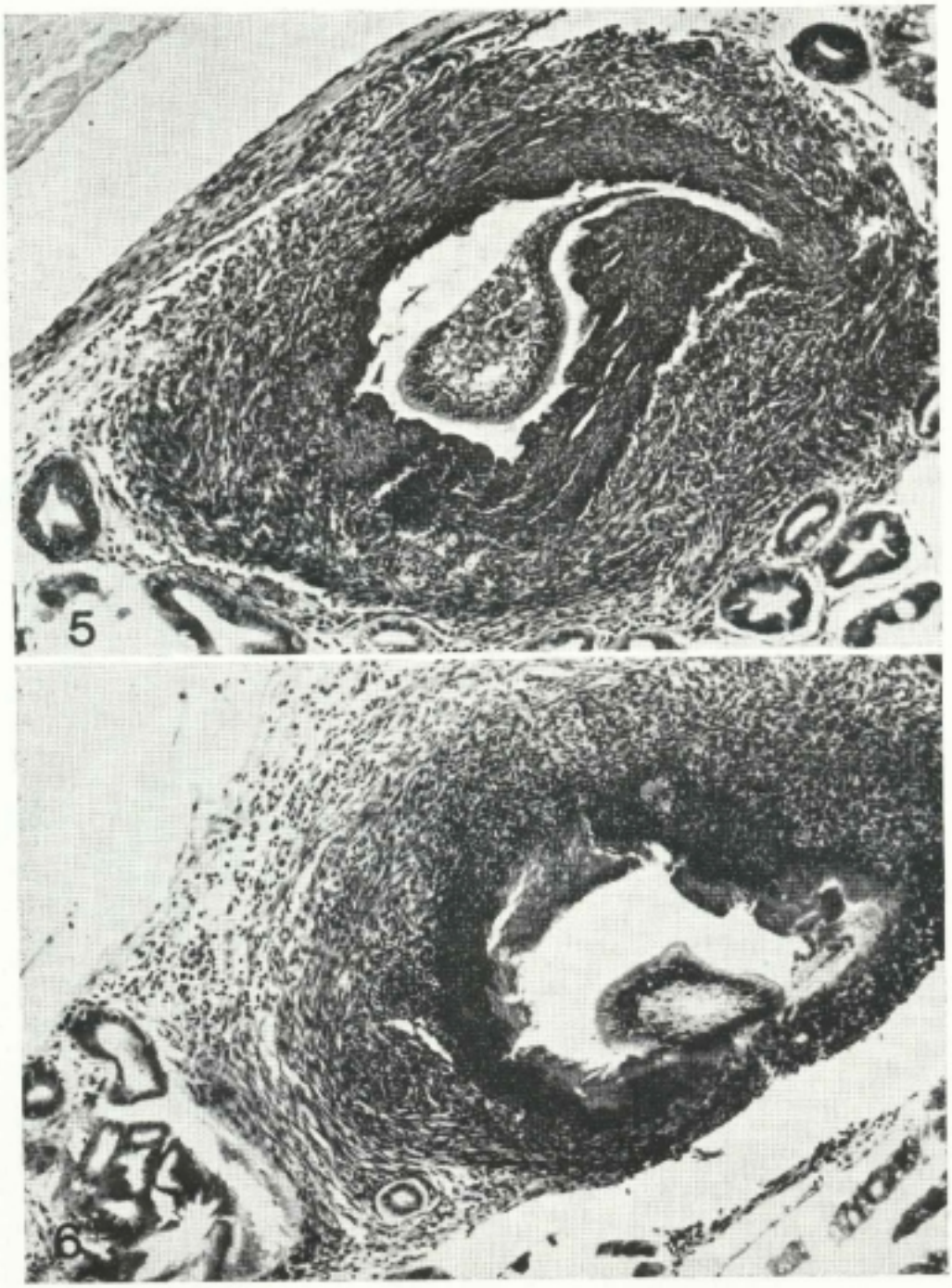


FIG. 5. Encapsulation which has progressed to a very thick-walled cyst interspersed with numerous, small fibers. The larva appears to be necrotic. H&E, ×175.

FIG. 6. Cyst in which the innermost hemocytes have become necrotic and melanization has occurred; the larva appears to be necrotic. Note the tubule which has been included in the cyst wall. H&E, ×220.

portion of the hepatopancreas (Fig. 1), and in the hemocoel immediately peripheral to the hepatopancreas and separated from that organ by the connective tissue sheath that surrounds the hepatopancreas (Fig. 2). The range of host response in the hepatopancreas is from a thin encapsulating cyst (Fig. 1) to complete encapsulation and eventual destruction and dissolution of the cestode, resulting in a dense fibrous capsule surrounding a central melanized homogeneous necrotic mass (Figs. 3–8).

The mature cyst (Fig. 3) consists of numerous hemocytes, fibroblasts, and small

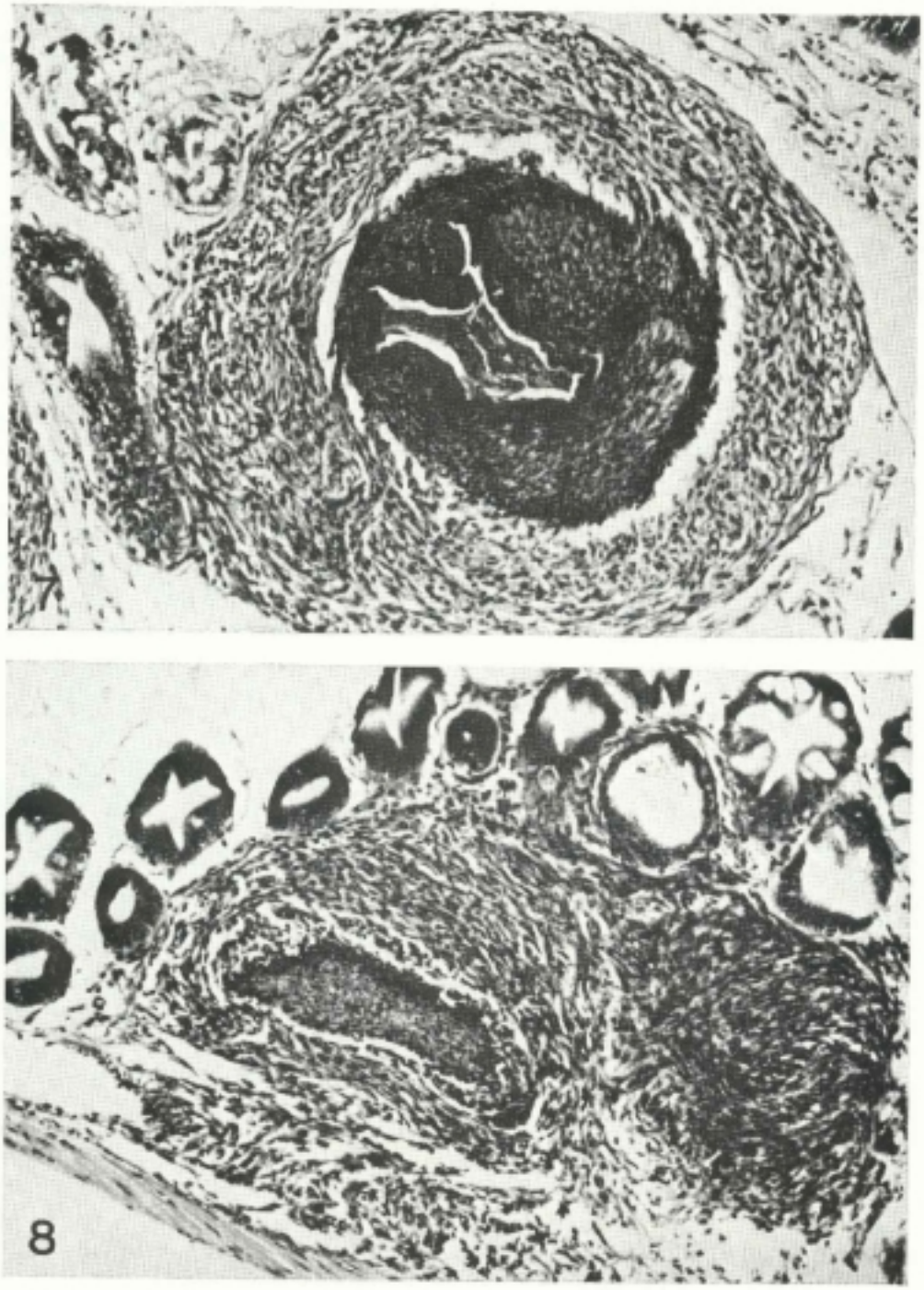
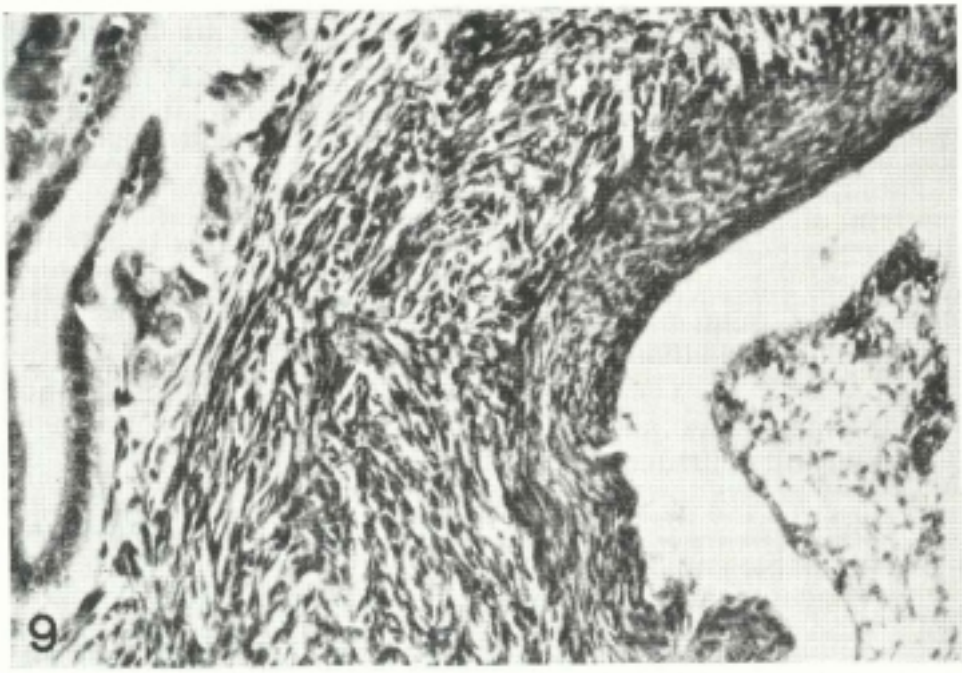


Fig. 7. Cyst which has progressed to a thick, dense, fibrous capsule with a dark, melanized inner nodule. The larva has approached complete destruction. H&E, ×220.

Fig. 8. A dense, fibrous capsule located between the acini of the outer portion of the hepatopancreas. No recognizable portions of the larva remain. H&E, ×220.

fibers (Fig. 9) which, when stained with Mallory's triple stain, may be interpreted to be collagen because of their staining reaction. The fibers are predominantly oriented parallel to the cyst wall and most of the hemocyte and fibroblast nuclei are elongated and oriented in the same plane (Fig. 9). The innermost layers of hemocytes are necrotic as evidenced by pyenotic nuclei and in some instances by cytolysis. These necrotic cells have become melanized, forming a thick brown inner nodule (Fig. 7). Tubules in the vicinity of a plerocercoid are crowded, reduced in size, stain much more



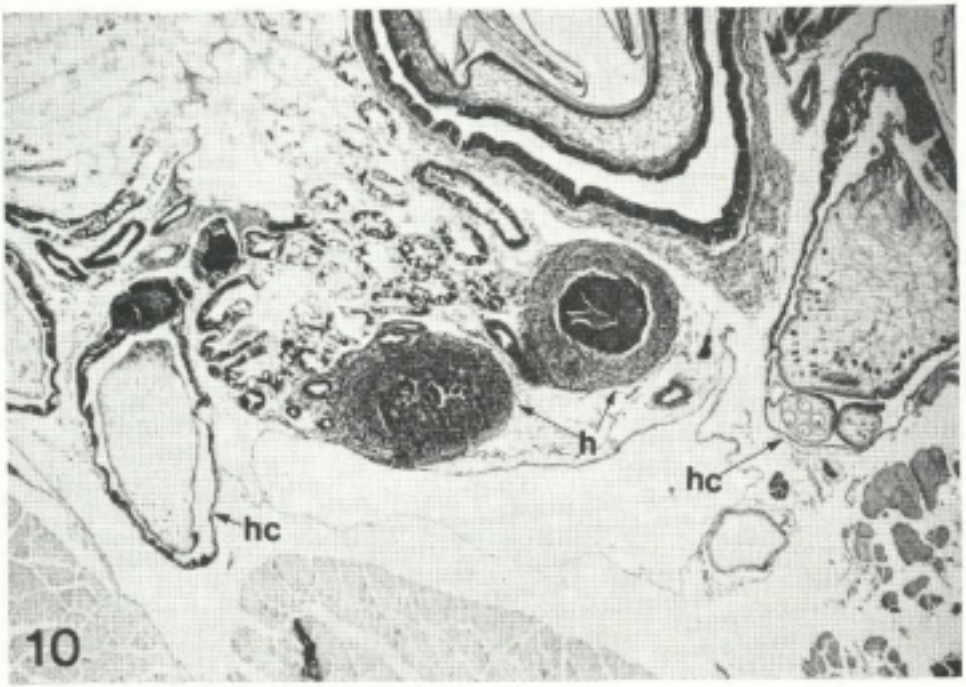


FIG. 9. The mature cyst wall consisting of numerous hemocytes, fibroblasts, and small fibers. Note the tubule, which appears to be flattened and reduced in size. H&E, ×440.

FIG. 10. The difference in intensity of host response to the plerocercoid larvae of *Prochristianella penaei* in the hepatopancreas and the hemocoel of *Penaeus setiferus*. H&E, ×145.

basophilic than normal, and are frequently incorporated into the cyst wall where they may be destroyed (Fig. 6).

Plerocercoids occurring in the hemocoel elicit a similar but much less intense response. A thin cyst wall, initially one or a few cells in thickness, is deposited around the cestode. Subsequently, the wall thickens, usually at the side of the cyst nearest the hepatopancreas, by emigration of hemocytes and fibroblasts to the area and deposition of collagenlike fibers. Melanization of hemocytes is intense in that portion of the cyst wall nearest the hepatopancreas

but rarely occurs in other locations. In no instances in our material, however, does the response proceed to destruction and resorption of the plerocercoid which occur in the hemocoel. Figure 10 clearly demonstrates the difference in intensity of host response in the hepatopancreas and the hemocoel.

Discussion

Although everyone who has examined *Prochristianella* in penaeid shrimp has apparently considered the hepatopancreas to be the normal location for the plerocercoid, the present study strongly indicates that the hemocoel is a more favorable site for survival of the cestode until eaten by the final host. Resorbing or even obviously moribund pleocercoids were not seen in the hemocoel, whereas almost every parasite within the hepatopancreas was heavily encapsulated and was frequently moribund or completely destroyed. We are convinced that the various stages of host response are clearly time related; thus the worm entering the hepatopancreas is quickly encapsulated, the cyst is progressively thickened until the plerocercoid is destroyed and eventually resorbed. This response in the shrimp is remarkably similar in some respects to that of the American oyster, *Crassostrea virginica,* to the metacestode of *Tylocephalum* as demonstrated by Cheng (1966) and Rifkin and Cheng (1968). Absent in the oyster's response but present in the shrimp's, however, are the numerous collagenlike fibers.

The resorption of the plerocercoids explains Aldrich's (1965) finding that after a rapid increase in intensity of infection (number of worms per host) in both brown and white shrimp with increasing size, the infective intensity leveled off in brown shrimp and declined in white shrimp. Aldrich proposed that this was due to a change in food habits or an increase in resistance. While these alternatives would account for the leveling off of intensity in brown shrimp, it cannot explain the decline in intensity of infection noted in white shrimp. If, however, the change in food habits occurred, with the larger juvenile shrimp no longer feeding on the small procercoid-infected invertebrates that are almost certainly the source of infection, the resorption process described would account for the decrease in intensity.

Investigation of the response of brown shrimp is planned. Aldrich (1965) reported that brown shrimp of any size almost always harbor more plerocercoids than white shrimp of comparable size. It may well be that brown shrimp are unable to destroy the plerocercoid as effectively as white shrimp.

Acknowledgments

The authors would like to express their appreciation to Mrs. Imogene Sanderson for preparing the slides and to Mr. Jeff Giles for collecting the shrimp.

References

Aldrich, D. V. 1965. Observations on the ecology and life cycle of *Prochristianella penaei* Kruse (Cestoda: Trypanorhyncha). *J. Parasitol.*, **51,** 370–376.

Cheng, T. C. 1966. The coracidium of the cestode *Tylocephalum* and the migration and fate of this parasite in the American oyster, *Crassostrea virginica. Trans. Amer. Microsc. Soc.,* **85,** 246–255.

Kruse, D. N. 1959. Parasites of the commercial shrimps, *Penaeus aztecus* Ives, *P. duorarum* Burkenroad and *P. setiferus* (Linnaeus). *Tulane Stud. Zool.,* **7,** 123–144.

Hutton, R. F., Sogandares-Bernal, F., Eldred, B., Ingle, R. M., and Woodburn, K. D. 1959. Investigations on the parasites and diseases of saltwater shrimp (Penaeidae) of sports and commercial importance to Florida. *Fla. State Bd. Conserv. Tech. Ser.,* **26,** 38 pp.

Rifkin, E., and Cheng, T. C. 1968. The origin, structure, and histochemical characterization of encapsulating cysts in the oyster *Crassostrea virginica* parasitized by the cestode *Tylocephalum* sp. *J. Invertebr. Pathol.,* **10,** 54–64.

Sparks, A. K., and Mackin, J. G. 1957. A larval trypanorhynchid cestode from commercial shrimp. *Tex. J. Sci.,* **9,** 475–476.

Woodburn, K. D., Eldred, B., Clark, E., Hutton, R. F., and Ingle, R. M. 1957. The live bait shrimp fishery of the west coast of Florida (Cedar Key to Naples). *Fla. State Bd. Conserv. Tech. Ser.,* **21,** 31 pp.